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Effects of Ambient Temperature and Exposure to 2450-MHz Microwave Radiation on Evaporative Heat Loss in the Mouse

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Effects of Ambient Temperature and Exposure to 2450-MHz Microwave Radiation on Evaporative Heat Loss in the Mouse

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ABSTRACT

Whole-body evaporative heat loss was measured as whole-body evaporative water loss in mice during a 90-min exposure to 2450-MHz microwave radiation at an ambient temperature of 20°C and in non-exposed mice maintained at ambient temperatures of 20, 25, 30, 33, and 35°C. The ambient-temperature threshold for increasing evaporative water loss was between 30 and 33°C. A specific absorption rate of microwave radiation in excess of 29 W/kg was required to produce an increase in heat loss. For absorption rates ranging from 29 to 44 W/kg, the mouse dissipated 65% of the total absorbed heat by water evaporation; the remainder was dissipated passively. The data collected in the mouse may be extrapolated to larger species, such as man, but only by an exponential relationship. Using this relationship, it was shown that a threshold specific absorption rate of 29 W/kg in a 0.033-kg mouse was equivalent to approximately 0.25 W/kg in a 70-kg human.

INTRODUCTION

The deep-body temperature of a mammal increases when it is exposed to high ambient temperatures [Adolph, 1947] or intense microwave exposure [Michaelson et al., 1961; Lu et al., 1977; Lotz and Michaelson, 1978]. Hyperthermia may not, by itself, be a sufficient end point for assessing the bioeffects of microwave radiation, since an elevation in temperature represents a point where the capacity of the regulatory system has been exceeded. In assessing the effects of ambient heat stress on the thermoregulatory system, investigators have measured end points more sensitive than hyperthermia, such as metabolism, evaporative heat or water loss (EHL or EWL), behavior, and peripheral vasomotor control [Bligh, 1973]. Only recently have some of these parameters, including behavior [Adair and Adams, 1980b], vasomotor control [Adair and Adams, 1980a], and metabolism [Ho and Edwards, 1977a,b] been measured in mammals exposed to microwaves.

Evaporative heat loss is a principal mechanism of heat dissipation during ambient heat stress [Bligh, 1973; Hardy and Bard, 1974]. Thermoregulation in hot environments is severely reduced in desalivated rats [Stricker and Hainsworth, 1971]. Both sweating (e.g., primates) and nonsweating mammals (e.g., rodents) increase EHL when exposed to high ambient temperatures [Getz, 1968; Hainsworth et al., 1968; Smiles et al., 1976]. Evaporative heat loss is an excellent parameter for detecting heat stress during microwave exposure because EHL is measured by withdrawing the expired air from the exposure chamber without disturbing the animal.

The purpose of this study was to assess the threshold specific absorption rate (SAR) of 2450-MHz microwaves required to increase EHL in the mouse. The threshold SAR determined in this study was compared with the SARs of other microwave-induced physiological effects in larger species, including man.

MATERIALS AND METHODS

Species

The animals used in this study were nine 4-month-old male mice, CBA/J strain, with a mean body weight of 33.7 ± 0.2 g (SE). Naïve mice normally take several hours to adjust to the novel

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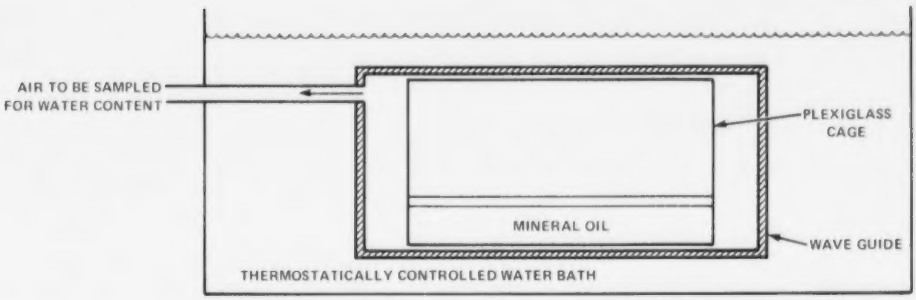

Figure 1 Cross-sectional view of the 2450-MHz waveguide exposure system. The dimensions of the circular plexiglass cage are 3.4 × 6.7 cm.

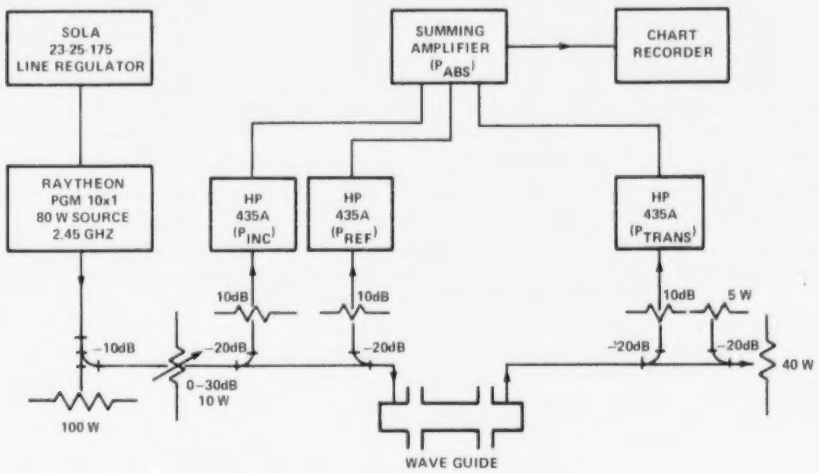

Figure 2 Schematic diagram of the pathway of microwave power through the waveguide system.

surroundings of the exposure chamber. Thus, the animals in this study were habituated to the exposure chamber by periodically exposing them to microwaves inside the waveguide at least once per week for a month. Since it was important to measure EHL in resting, non-agitated mice, the same animals were used over the 38 days of experimentation. The animals were maintained at 22°C under a 12:12 L:D photoperiod. (Lights were turned on at 6 a.m.)

Apparatus

Whole-body EWL was measured in mice exposed individually to 2450-MHz radiation inside a rectangular waveguide, 5.8 × 11.2 × 30.5 cm (Fig. 1). The entire waveguide was immersed in a water bath thermostabilized to ±0.5°C. The water-immersed system allowed for a uniform control of air and wall temperatures inside the waveguide. Air from the waveguide was drawn through a cannister filled with color-indicating anhydrous calcium sulfate at a flow rate of 60 ml/min. Ambient air dried in calcium sulfate was passed into the waveguide via a length of copper tubing equilibrated with the temperature of the water bath. The increase in weight of the anhydrate cannister filtering the exhaust air, determined to the nearest milligram, was taken as the amount of whole-body EWL, or EHL, over a specified time of exposure to temperature or microwaves.

Continuous-wave 2450-MHz microwaves were generated with a Raytheon radiator (PGM-

10X1). The radiation passed through a Narda bidirectional coupler (No. 3022) into the waveguide, then into another bidirectional coupler (Fig. 2). The couplers allowed for monitoring the incident, reflected, and transmitted power using Hewlett-Packard power meters (Model No. 435A). The amount of power absorbed by the mouse was determined by summing the three analog voltage outputs (absorbed = incident − reflected − transmitted) from the power meters. A detailed description of the waveguide system has been given by Ho and Edwards [1977a,b]. For an object the size of a mouse, 2450 MHz is the resonant wavelength [Durney et al., 1978].

Protocol

During microwave and ambient temperature exposure, the mice were allowed free movement inside a perforated, cylindrical plexiglass cage (3.4 × 6.9 cm). A layer of mineral oil was placed beneath to trap urinary and fecal moisture, and to separate this content from the final determination of EHL. Varying the volume of mineral oil did not affect power absorption. The mouse was placed in the waveguide and allowed to equilibrate for 90 min. Microwave exposures were made at incident powers of approximately 0.25 to 2.75 W for 90 min at an ambient (water-bath) temperature of 20°C. The intensity of microwave exposure was selected at random.

Evaporative heat loss was also measured in single mice maintained at ambient temperatures of 20, 25, 30, 33, and 35°C. A 90-min adjustment period at a particular ambient temperature was followed by a 90-min measurement of EHL. The mice were allowed at least 48 h for recovery before being subjected to another microwave or ambient-temperature treatment.

Analysis of Data

Evaporative heat loss (W/kg) was calculated by multiplying the whole-body EWL (mg H_2O/g/s) by 2.426 J/mg H_2O (heat removed by evaporating 1 mg of water at a temperature of 37°C [see Kleiber, 1975]). The absorbed microwave power recorded was integrated with planimetry to obtain a mean SAR (W/kg) over the 90-min exposure period. The data were analyzed with linear regression statistics.

RESULTS AND DISCUSSION

At an ambient temperature of 20°C, whole-body EHL was relatively constant between SARs ranging from 0 to 29 W/kg (Fig. 3). Above 29 W/kg, the mouse underwent a linear elevation in EHL with increasing SAR. The slope of the regression line (see Fig. 2) — 0.65 W/kg evaporated heat per W/kg absorbed heat — implies that 65% of the absorbed microwave energy was dissipated by evaporation while the remaining 35% was dissipated passively.

Absorbed power was relatively constant at low incident powers but tended to fluctuate at the high power range (Fig. 4). Presumably, the fluctuations resulted from the animal's changing its position in the waveguide during radiation exposure. The extensive movement of the animal at the high incident powers was interpreted as an indication of discomfort due to the high amount of absorbed energy [Rugh, 1973].

Apparently, these data are the first to relate the effects of ambient temperature and microwave exposure to the activity of physiologic heat dissipation. When mice were maintained at an ambient temperature of 20°C, an SAR of 29 W/kg was required to significantly raise EHL (the microwave heat

Table 1 Mean ± standard error of evaporative water loss and evaporative heat loss in mice maintained at ambient temperatures of 20 to 35°C

Ambient temperature (0°C)	Evaporative water loss (mg/g/min)	Evaporative heat loss (W/kg)
20	0.062 ± 0.013	2.49 ± 0.52
25	0.052 ± 0.004	2.10 ± 0.16
30	0.05 ± 0.004	2.02 ± 0.16
33	0.15 ± 0.037*	6.06 ± 1.49
35	0.13 ± 0.004**	5.25 ± 0.16

*$p < 0.025$ when compared to 30°C group.
**$p < 0.005$ when compared to 30°C group.

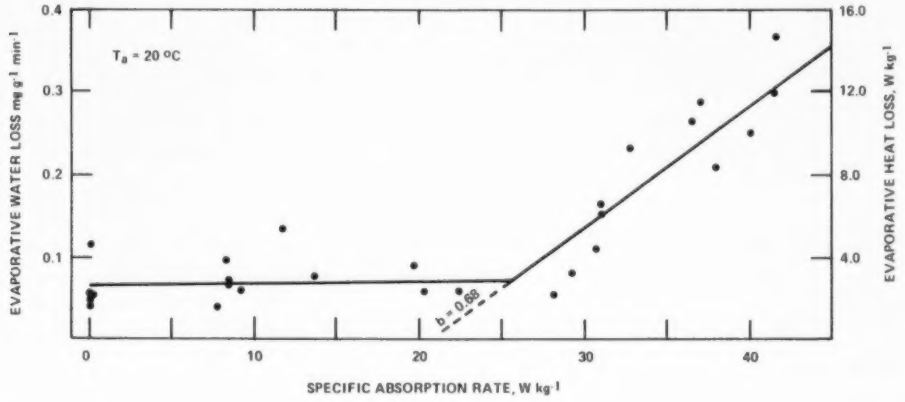

Figure 3 Relationship between EHL and SAR in mice exposed to 2450-MHz microwave radiation. Each point represents one 90-min determination of EHL at an average SAR. $N = 27$ data points collected in 9 animals. $r = 0.9159$ for the regression line above 29 W/kg.

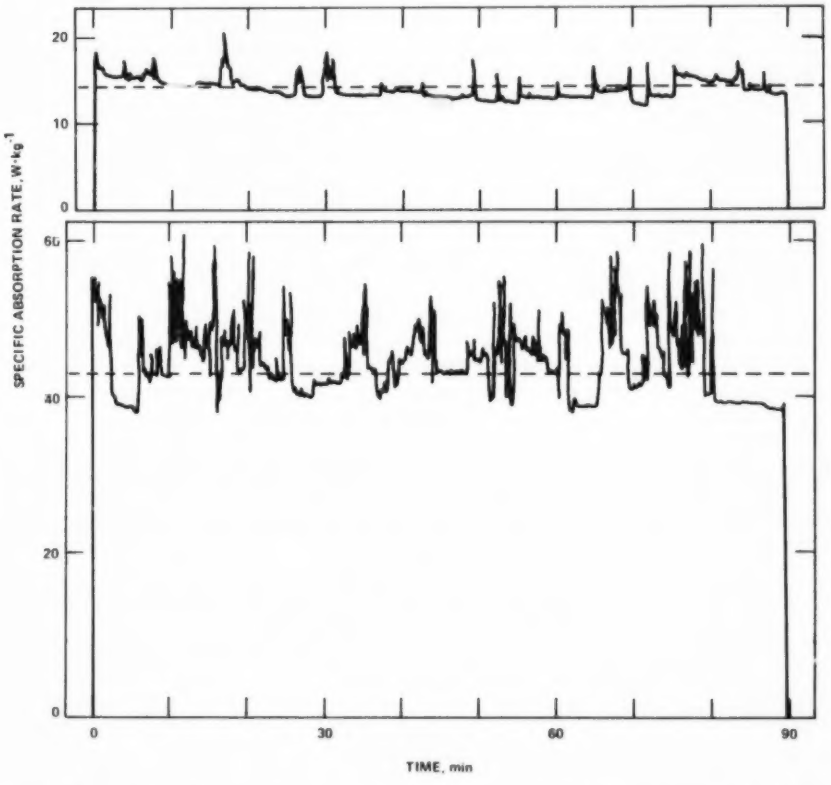

Figure 4 Time course of power absorption during exposure to 2450-MHz microwave radiation at low (1.0 W) and high (2.75 W) incident powers. Broken line equals the average SAR as determined by integrating the absorbed power curve with planimetry. Note large fluctuations in absorbed power when the mouse is exposed to a relatively high incident power.

load was equivalent to approximately 290% of the resting metabolic heat production of the mouse). That EHL did not increase below 29 W/kg indicates that thermal homeostasis can be maintained by passive dissipation of the entire absorbed microwave energy, mainly through radiative and convective heat loss. The threshold EHL of 29 W/kg at 20°C suggests that an SAR range of 0−29 W/kg may be thermally equivalent to an ambient temperature range of 20−30°C.

Evaporative heat loss remained stable between ambient temperatures of 20 and 30°C (Table 1). Increasing ambient temperature to 33 and 35°C resulted in an approximate doubling of resting EHL.

A threshold of 29 W/kg may appear to be an excessive amount of absorbed heat; however, it is important to consider the size of the animals used in this study (~ 0.034 kg). The relatively small mass of the mouse provides the animal with a large surface-area-to-body-volume ratio. The greater the surface/volume ratio, the faster the rate of heat loss per unit body weight. This ratio is critical in determining the ability of an animal to dissipate a heat load passively rather than using active physiologic heat dissipation (EHL). Furthermore, because surface/volume ratio correlates inversely to volume and body mass, small mammals must, to maintain a constant body temperature, generate more heat per unit body weight than larger species. Thus, under resting conditions, the metabolic rate of a 0.03-kg mouse is approximately 7.3 × that of a 70-kg man [Schmidt-Nielsen, 1975].

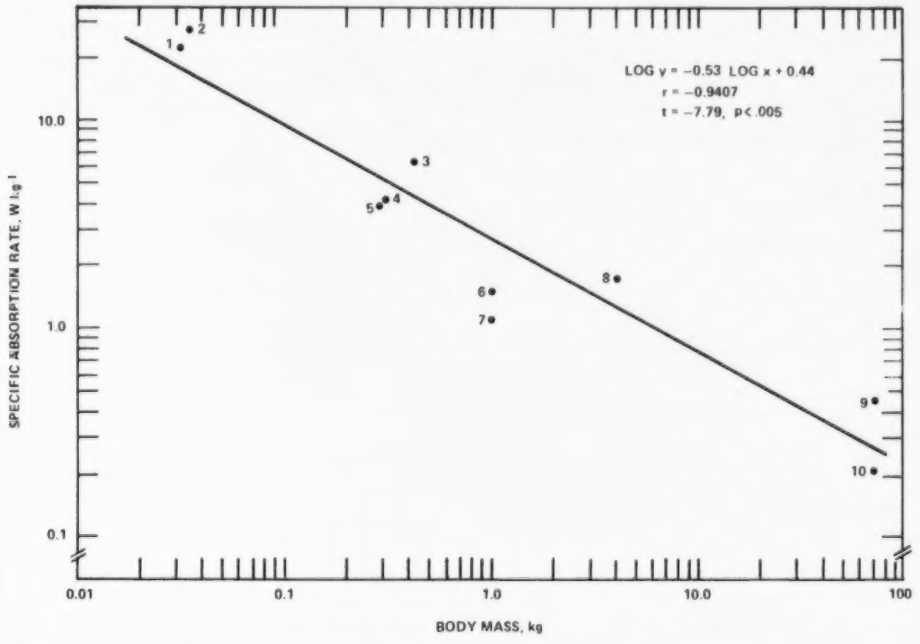

Figure 5 Relationship of body mass to the amount of absorbed heat necessary to alter the activity of various physiological systems. Each datum represents the minimal SAR necessary to alter a physiological response. Ambient temperature in these studies ranged from 20 to 30°C. The number adjacent to each data point corresponds to the species, physiological parameter, frequency, and literature reference, respectively:
(1) Mouse; inhibit metabolism; 2450 MHz; Ho and Edwards, 1977b.
(2) Mouse; increase EHL; 2450 MHz; Gordon, present study.
(3) Rat; inhibit metabolism; 2450 MHz; Phillips et al., 1975.
(4) Rat; increase serum corticosterone; 2450 MHz; Lu et al., 1981.
(5) Rat; increase serum corticosterone; 2450 MHz; Lotz and Michaelson, 1978.
(6) Squirrel monkey; increase skin blood flow; 2450 MHz; Adair and Adams, 1980a.
(7) Modify thermoregulatory behavior; 2450 MHz; Adair and Adams, 1980b.
(8) Rabbit; raise heart rate; 2450 MHz; Birenbaum et al., 1975.
(9) Man (model); increase EHL; 200 MHz; Spiegel et al., 1980.
(10) Man (model); increase EHL; 80 MHz; Spiegel et al., 1979.

There appear to be no data on record relating the SAR from microwave exposure required to alter the activity of a physiological system in humans. Unless it is possible to collect human data, it will be necessary either to model human thermoregulatory responses or to extrapolate data collected in laboratory mammals to man. As regards the latter approach, the question arises: Should an extrapolation of the bioeffect data from laboratory mammals to man be linear or exponential? For example, would the threshold of 29 W/kg for activating EHL in the mouse be the same for a 70-kg human?

Data from various studies showing the minimal SAR necessary to affect a physiological parameter in mammals of various weights are plotted in Fig. 5. In spite of the wide variation in ambient temperature, microwave frequency, species, and physiological parameter, the strong statistical significance of the regression line ($r = -0.9407$, $p < 0.005$) implies that body mass has a critical function in determining threshold SAR. Note that the best fitting linear regression is a double logarithmic correlation; hence, extrapolation of a threshold SAR measured in a small laboratory mammal necessitates a logarithmic scaling factor. For example, a threshold SAR in a 0.034-kg mouse of 29 W/kg is predicted to be equivalent to an SAR of 0.25 W/kg in a 70-kg man.

The data in Fig. 5 suggest that SAR-bioeffect data collected in rodents can be extrapolated to humans, but only in a logarithmic manner. For example, under thermoneutral conditions an SAR of ~0.25 W/kg (20% of the metabolic rate) is predicted to be an adequate stimulus to alter the activity of various physiological systems in a 70-kg human. It is important to note that Fig. 5 was constructed from studies performed in an array of laboratories using different exposure techniques and environmental conditions. Variables such as physiological parameter, ambient temperature, humidity, and age, to name a few, should affect the slope and intercept of the regression line. It will be of great interest to develop correlations between SAR and body mass with the measurement of a single physiological parameter under specific environmental conditions.

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